



International Journal of Current Research Vol. 6, Issue, 11, pp.9778-9780, November, 2014

## **CASE STUDY**

# TETANUS: FOLLOWING TONGUE LACERATION

# \*Thaer Hameed Mohsin

Head of Oral and Maxillofacial Surgery Department, Collage of Dentistry, Wasit University, Iraq

#### ARTICLE INFO

#### Article History:

Received 15<sup>th</sup> August, 2014 Received in revised form 26<sup>th</sup> September, 2014 Accepted 04<sup>th</sup> October, 2014 Published online 18<sup>th</sup> November, 2014

#### **ABSTRACT**

Tetanus adisease caused by infection by an aerobic bacteria called closteridum tetanae its present mainly in the soil and growth in anaerobic field, in this literature A case of tetanus is described in a 43 years old Male in AL-kindy educated hospital, Baghdad, Iraq. The initial presentation was Trismus and dysphagia following a tongue laceration by grossly carious tooth, Subsequently he developed muscular spasm which led to the diagnosis of tetanus. This paper discusses the general management of tetanus

#### Key words:

Tetanus, Clostridium tetane tongue laceration, Jaw spasm

Copyright © 2014 Thaer, Hameed Mohsin. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

# INTRODUCTION

Tetanus is a disease not commonly seen in Iraq; it is a rare case due to the active immunization program and routine administration of tetanus toxoid. In case of trauma. Clostridium tetani a (gram positive bacillus) is a commensally organism found in the guts of man and domestic animals as well as in cultivated soils. It is strict an aerobic which forms spores which characteristic "Dum stick appearance (1). Reduction in oxygenation is essential for its germination, the incubation period various from 3 days to 4 week. The shorter the period the worse the prognosis in 60% the portal of entry of the bacterium is a wound often of arrival nature and history of injury may be difficult to elicit. no obvious entry site can be found (2) Tetanus may be present years after the injury the bacteria produce anaerobic toxin, tetano spasmin 'as well as (aheamolysin) the toxin which effect the CNS is powerful poison, second only to the botulinus toxin in its potency and its main action is at the interneuronal synapse of the inhibitory path ways where it produce a blockade of spinal inhibition resulting in muscular rigidity (3) the muscles most affected are those with short motor neurons, such as those found in the head and neck region the toxin having no effect on sensory nerves.

# Clinical feature

The disease is commonly present with pain and stiffness of the jaw, neck and back musculature. The patient shows early trismus due to massetric spasm with hyper tonicity of the muscle in the neck, back, abdomen and limb and spasm of the

\*Corresponding author: Thaer, Hameed Mohsin,

Head of Oral and Maxillofacial Surgery Department, Collage of Dentistry, Wasit University, Iraq.

facial muscles. Classically there are two presentations, namely, cephalic tetanus and local tetanus; in the former the infection arises in the head and neck region, and is associated with individual, cranial nerve palsies, notably unilateral seventh nerve lesion and involvement of the ocular muscle resulting in diplopia (4). The interval between the onset of symptoms and the muscle spasm varies from 24 hours to ten days; the shorter interval the more sever the tetanus and worse the prognosis tetanus which develop in wound of the head and neck region is said to be more frequently fatal than from infection in the lower part of the body (5). The significant morbidity and mortality of disease are duo to complication such as in adequate ventilation after over activity of the sympathetic nervous system

## Differential diagnosis

Other conditions present with trisms must be excluded particularly local infection associated with dental and adjacent tissues, tempromandibular joint dysfunction, meningitis, encephalitis, hysteria, drug induced dyskinaesias, such as those associated with metoclopramide or perphenazine (6). The diagnosis of the condition is entirely clinical, clostridium tetani being found in only 30%, not infrequently the organism is found in patients not suffering from the disease (7)

### Treatment and prophylaxis

An active immunization program makes this a preventable disease. Generally the disease must be treated by elimination of the organism neutralization of the toxin and control of the muscular spasm along with the symptomatic treatment of the respiratory system and cardio vascular system (8). Prophylaxis

is achieved by active immunization which in childhood starts with injection of a triple vaccine [Diphtheria, pertussis, tetanus antigens] The first injection is given at 12 week of age, the second at 18 to 20 weeks and the final at six months. Immunity is said to last 10 years, booster injection being given at five to ten years interval (9). Human anti tetanus immunoglobulin [ATGHumotet] is available and to be effective must be given early in the course of the disease. It confers protection for at least four weeks (10) but is ineffective after the toxin becomes bound to nervous tissue.

### Case report

A 43 years old male presented to the Maxillofacial Department in Al-Kindy teaching hospital, Baghdad, Iraq, with trismus and ataxia. History of biting his tongue one week earlier was obtained three days ago (Figure 1) after this incident he noted



Figure 1. Laceration (chronic tongue bite)

difficulty in jaw opening on examination he was found he have gross caries sever periodontal involvement and complained of dysphagia which he felt duo to a sore throat during clinical examination by physician, he was found to have tacky cardia [pulse rate80] blood pressure 120/90. His chest was clear and in the neuromuscular examination, the power, tone, co ordination and reflexes were unremarkable during laboratory investigation [biochemical and hematology] show dehydration, hypernateraemia, hyperkalemia and white cell count increased predominantly neutrophils. An orthopantomograph revealed that dental infection may be the cause of the trismus after 2days of admission the patient develop un explain extension spasm during this spasm the patient became short of breathing and cyanosed this muscle stiffness became more generalized and involved the jaw and neck musculature.

tetanus was diagnosis on clinical ground and treatment in the intensive care unit was investigated using human antitetanas globulin 500I.U. Intra muscular start with benzyl penicillin one mega unite six hourly and diazepam 5milligram intravenously four hourly, it was decided to in tubate and ventilate the patient as part of his primary care, initially the patient condition deteriorate, the muscle rigidity increase, stimulated some spasms lasting from two to three minute he show symptoms of bulbar involvement with complete ophthalmoplegia [cephalic tetanus] the hemoglobin fell from 14.0 to8.0g/dl and the potassium was raised to 7.0mmol/1[normal 3.5-5.0mmol/1]. The clinical picture of establish tetanus was now apparent and

therefore tracheotomy was performed and the patient placed on continuous monitoring for three weeks.

### DISCUSSION

It was felt in the case of tetanus described, that the cause was the tongue being lacerated by aseptic dentition tetanus has been described in associated with dental procedures and oral sepsis, the condition may present to the dental surgeon in the first instance since trismus is often the first symptom (11). It is important to consider that trismus may be caused not only by local sepsis but by tetanus as well .this patient had never been immunized, his percentage was classical when viewed in retrospect, having presented with trismus and dysphagia (12). Treatment of the condition depends on severity of the symptoms. Generally, one must control the muscle spasm, maintain an air way (13).

Grading treatment and results believed that in the mild form of disease where no dysphasia or respiratory problems existed. diazepam was of help in relieving the symptoms of spasticity and trismus, in the moderate type dysphagia, respiratory difficulty, muscle spasm occur, and his tracheasotomy and diazepam are recommended. In the severe form of the disease neuromuscular blockade and artificial ventilation are deemed essential, the very severe form show labile hypertension, tachycardia, ECG abnormalities, vasoconstriction profuse sweating and pyrexia, all symptoms believed to be due to sympathetic stimulation (3). Here recommended heavy sedation and interments of a beta- blocker (8). Careful monitoring of the heart rate as an indicator as when to use the blocking agents (5). feels that the autonomic effects can be controlled with alpha and beta blocker in the form of labetalol. The severity of tetanus is inversely proportional to its incubation period and should the time of onset of the first symptoms be less than 48 hours it is regarded as a poor prognostic sign, mortality even with the best pharmacological and supportive care can vary from nil to greater than 60%. (7) Reported in his series amorality in the region of ten percent. Tetanus resulting from dental sepsis is rare, the case described may alert the profession to the possible sequel of aseptic dental condition illustrating some of the difficulties encountered in its diagnosis and management and gives support to the need for immunization against the condition

# **REFERENCES**

- 1. Cawson 1998. Essential of oral pathology and oral medicine Cook, T M. Tetanus: Reviews of the literature 2001. *Br J of anesthesia*, "6":67-69
- Hinman, A. and Orenstein 1999. W. Public health considerations. 1085-88
- Walter and Israel, 1981. Principle of pathology for dental Student: 120-125
- 4. Meauder E., Gpenod, 2005. mandibular nerve block for the removal of denture during trismus caused by tetanus, *Anasthesia and Analgesic Journal* "1":23-25
- 5. Microbiology and pathology-infection control today 2004. Clinical update by Marcia Hardwick clostridium edificial toxin, diagnosis, treatment:56-58

- Neville, 1995. Oral and maxillofacial pathology:220-222 complication of tetanus Geoffrey L Howe 1985, minor oral surgery:230-233.
- 7. Sanders RKM. 1996. Management of tetanus trope doctor, vol 26:122-124.
- 8. Slifkin R T,Clark, S.J., Strandhoy, S.E., Konrad, T.R. 1997. Public-sector immunization coverage in 11states: The status of rural areas. *J. of Rural Health*, "4":334-341.
- Stokley, S, Smith, P.J, Klevens, R.M., and Battaglia 2001.
  M.P. Vaccination status of children living inrural areas in the United States: are they protected *American Journal of Preventive Medicine*, 20Supplement):55-60.
- 10. Tetanus encyclopedia Britannica 2007. Encyclopaedia Britannica on line:22-24
- 11. Thwaites Cl and J Jfarrar, 2003. BMJ, preventing and treating tetanus"23"134-135
- 12. Mcook T., R. Tprotheroeand J.M handel. 2001. *Br.J. Anesthesia*, "3":24-27
- 13. Wilson ME. 1991. A word guide to infection, disease distribution, diagnosis:243-245

\*\*\*\*\*